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CLINICAL STUDIES

# Determinants of Left Ventricular Aneurysm Formation After Anterior Myocardial Infarction: A Clinical and Angiographic Study

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**To determine factors involved in left ventricular aneurysm formation after transmural anterior myocardial infarction, 79 patients with a first myocardial infarction who underwent cardiac catheterization within 6 months of infarction were evaluated. Patients who had received thrombolytic therapy were excluded. Patients were divided into four groups depending on the status of the left anterior descending artery and the presence or absence of a left ventricular aneurysm: Group I (n = 25): aneurysm with occluded left anterior descending artery; Group II (n = 27): no aneurysm and occluded left anterior descending artery; Group III (n = 23): no aneurysm and patent left anterior descending artery; and Group IV (n = 4): aneurysm with patent left anterior descending artery.**

**Single vessel disease was more common in Group I (aneurysm) compared with Groups II and III (no aneurysm) ($\chi^2_4$ = 12.8; probability value equal to 0.012). Collateral blood supply in the presence of an occluded left anterior descending artery was significantly less in Group I (aneurysm) compared with Group II (no aneurysm) (0.9 versus 2.4, $p < 0.001$). The extent of coronary artery disease and collateral blood supply in Groups I and II were directly related ($p = 0.012$). Neither age, sex nor risk factors for coronary disease correlated with aneurysm formation. At a mean follow-up of 48 months, no differences were observed in the incidence of recurrent angina, new myocardial infarction, embolic events or sudden death. More patients in Group II underwent coronary artery bypass surgery.**

**Total occlusion of the left anterior descending artery in association with inherent poor collateral blood supply is a significant determinant of aneurysm formation after anterior myocardial infarction. Multivessel disease with either good collateral circulation or a patent left anterior descending artery is uncommonly associated with the development of left ventricular aneurysm.**

***(J Am Coll Cardiol 1986;8:1256–62)***

Left ventricular aneurysm formation may complicate transmural myocardial infarction (1–8); 7.6% of 15,019 patients in the Coronary Artery Surgery Study (CASS) study developed an aneurysm (1). Left venticular aneurysm is more frequent after anterior myocardial infarction and in patients without a prior history of angina pectoris (2,3,5,8). The mortality rate is increased in these patients, probably related to the amount of left ventricular dysfunction (9,10). However, the exact pathogenesis of left ventricular aneurysm formation complicating anterior myocardial infarction remains unclear. Regional thinning of necrotic myocardium (expansion) has been proposed as a cause of aneurysm formation (11,12). The relation between the extent of coronary disease and aneurysm formation remains controversial (1–6), and the role of collateral blood supply to the ischemic zone has not been systematically studied. Therefore, we retrospectively examined clinical and angiographic variables that might play a role in the development of a left ventricular aneurysm in patients after a first transmural anterior myocardial infarction.

## Methods

**Selection of patients.** A retrospective analysis was performed involving 10,066 patients who had undergone cardiac catheterization between 1975 and 1985 at the Vanderbilt University Medical Center and Nashville Veterans Hospital. Only patients who underwent cardiac catheteriza-

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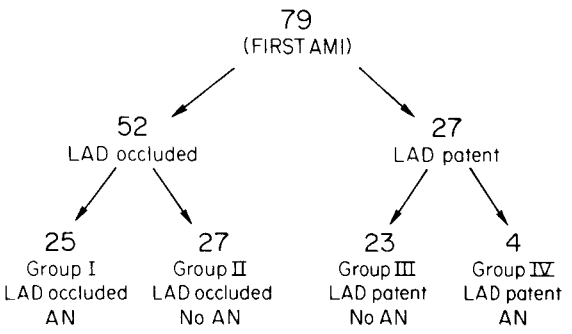



**Figure 1.** Diagrammatic representation of patient population. The 79 patients were grouped depending on the status of the left anterior descending (LAD) artery and the presence or absence of a left ventricular aneurysm (AN). AMI = anterior myocardial infarction.

tion within 6 months of infarction were included in the study. All patients had an extensive transmural anterior myocardial infarction as assessed by electrocardiographic criteria (development of q waves in leads I, aVL, $V_1$ to $V_5$ or $V_6$). Patients with a documented prior myocardial infarction or those treated with thrombolytic agents were excluded.

Seventy-nine patients who met these defined criteria were included in the final analysis (Fig. 1). Cardiac catheterization was performed a mean of 2.3 ± 0.8 months after infarction. These patients were categorized on the basis of 1) the status of the left anterior descending coronary artery, and 2) the presence or absence of a left ventricular aneurysm: Group I (n = 25): aneurysm with occluded left anterior descending artery; Group II (n = 27): no aneurysm and occluded left anterior descending artery; Group III (n = 23): no aneurysm and patent left anterior descending artery; and Group IV (n = 4): aneurysm and a patent left anterior descending artery.

**Clinical features.** The clinical features analyzed included age, sex, and risk factors for coronary artery disease (cigarette smoking, diabetes mellitus, hypertension, family history of infarction at less than 50 years of age and total serum cholesterol). Killip functional class at the time of infarction and prior treatment with antianginal medications (in particular, beta-adrenergic blocking agents) were also noted. Clinical outcome was assessed by review of medical records and telephone interviews with the patient or his or her physician. Recurrent angina pectoris, new myocardial infarction, embolic events, ventricular tachycardia, congestive heart failure, coronary artery bypass surgery and mortality were noted.

**Angiographic analysis.** Angiography was performed using the Judkins technique and recorded on 35 mm cine film at 30 frames/s. Left ventricular end-diastolic pressure was measured before ventriculography which was performed in the 30° right anterior oblique projection. Selected coronary angiography was performed in multiple standard and angulated views for optimal visualization of stenotic segments.

**Grading of coronary artery disease (Fig. 2).** The extent of coronary artery disease was assessed by two independent angiographers using a caliper method, and this measurement, collateral blood supply score and segmental wall motion abnormalities had a 95% interobserver agreement. When differences existed, they were resolved by a third reviewer or by concensus. Patients were categorized as having single, double or triple vessel disease when a significant lesion (defined as greater than 50% narrowing of intraluminal diameter) was present in one or more coronary arteries or a major branch, or both (9,13). The extent of coronary

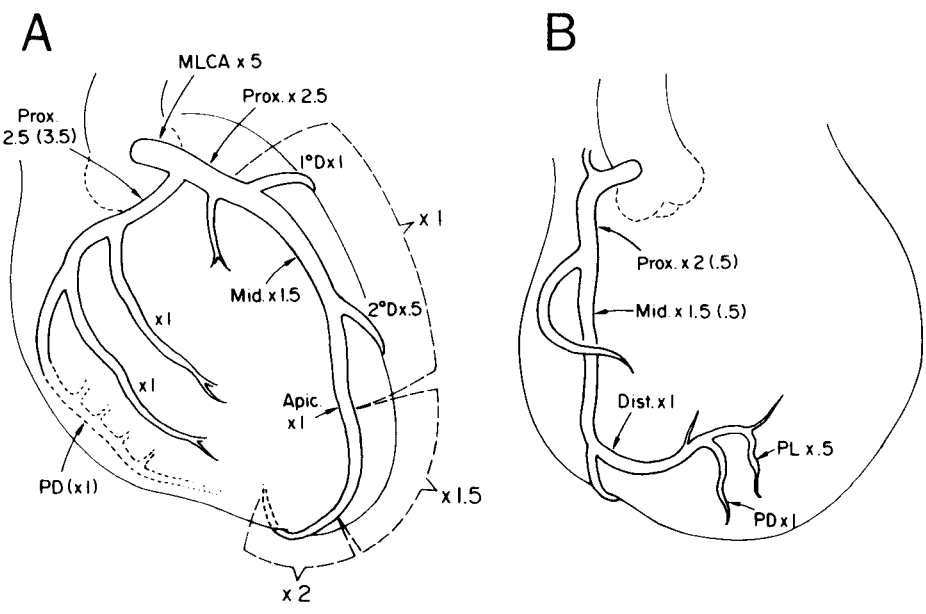



**Figure 2.** Grading of the severity of coronary artery disease based on the system of Gensini (14). Each segment of the left (**A**) or right (**B**) coronary artery is followed by a multiplying factor depending on the functional significance of the area supplied by the vessel. An additional weighting factor was also utilized to account for the variable length of the left anterior descending artery; for example, if the vessel supplied the inferior wall, the score was multiplied by 2. Apic. = apical segment; 1° and 2° D = first and second diagonal branches, respectively; Dist. = distal segments; Mid. = middle segment; MLCA = main left coronary artery; PD = posterior descending artery; PL = posterolateral branch; Prox. = proximal segment.

disease was further analyzed using a modified system, based on that of Gensini (14) (Fig. 2), that took into account both the site of narrowing and the percent reduction in intraluminal diameter. A progressively greater reduction in luminal diameter received a proportionately higher score. For example, a score of 1 was given for a 25% obstruction and 32 for total obstruction. A weighting factor was then added to account for the site of obstruction; the more proximal a lesion, the higher the score (unmodified Gensini). Because the amount of myocardium supplied by the left anterior descending artery varies, an additional weighting factor was also utilized (modified Gensini). For example, in patients with a large left anterior descending artery that supplied the inferior wall the total left anterior descending score was multiplied by a factor of 2.

**Collateral blood supply.** Collateral blood supply was graded on a scale of 0 to 3 depending on the degree of opacification of the occluded vessel. The score was based on the injection that best opacified the occluded vessel: 0 = no visualized collateral vessels; 1 = faint visualization of the occluded vessel; 2 = distinct opacification of the occluded vessel but less than the nonoccluded segment; and 3 = opacification of the occluded vessel to the same degree as the nonoccluded segment.

**Left ventriculography.** A left ventricular aneurysm was diagnosed angiographically using the CASS protocol (1). An aneurysm was said to be present when two of the following angiographic criteria were present: 1) systolic bulging of the involved segment, 2) absence of trabeculation in the involved segment, and 3) well defined demarcation of the infarcted segment from normally contracting myocardium (1). Most angiograms (92%) satisfied all three criteria. Left ventricular ejection fraction was calculated using the area-length method of Sandler and Dodge (15). Percent radial shortening in the ischemic zone was analyzed with a computerized program as previously described (16). Briefly, a longitudinal axis was constructed by connecting the middle of the aortic valve plane with the apex for both end-diastole and end-systole: Radii were then constructed at 10° intervals from the epicenter of the two silhouettes. Radial shortening in each radius was derived according to the formula: percent radial shortening = (end-diastolic length − end systolic length)/end-diastolic length × 100. Radii that involved the regions of the mitral and aortic valves were excluded (31 to 36 and 1 to 3, respectively). The ischemic zone was defined as less than 10% radial shortening and the nonischemic zone as greater than 10% radial shortening.

**Statistical analysis.** The data were analyzed using a VAX 11/780 computer utilizing the BMDP and SPSS$^x$ statistical packages. When analyzing the correlation between two discrete variables, chi-square analysis was used where appropriate. When the expected number in a given cell was less than 5, Fisher's exact test was utilized. Relations between pairs of continuous variables were analyzed using the Pearson Product Moment correlation. Finally, to determine the independent effects of the dependent variables when comparing pairs of groups, multiple logistic regression analysis was utilized. The advantage of using multiple logistic regression is that both continuous and discrete variables can be entered into the model simultaneously and it is possible to determine the effect of each variable adjusted for all other variables in the model. Data are presented as mean ± standard deviation.

## Results

**Clinical features (Table 1).** No significant differences were observed in age, sex or risk factors for coronary artery disease among the four groups. Total serum cholesterol was significantly greater in patients with an occluded left anterior descending artery and no aneurysm (Group II) compared with patients with an aneurysm (Group I) (232 ± 47 versus 192 ± 47 mg/%, $p < 0.01$). Time to cardiac catheterization was similar in all four groups.

**Angiographic features (Tables 2 and 3, Fig. 3 and 4).** Single vessel coronary disease was the most prevalent finding in patients with an aneurysm and an occluded left anterior descending artery (Group I). The extent of coronary artery disease was evenly distributed in Group II (occluded left anterior descending artery and no aneurysm). Patients with a patent left anterior descending artery and no aneurysm (Group III) tended to have more extensive coronary disease.

**Table 1.** Clinical and Biochemical Variables in the 79 Patients

| Group | Age (yr) | No. of Men | Interval From Catheterization to MI (mo) | SM | SH | FH | DM | TC (mg/100 ml) |
|---|---|---|---|---|---|---|---|---|
| I (n = 24) | 50.6 ± 11.8 | 20 | 2.4 ± 1.9 | 19 | 11 | 14 | 2 | 192 ± 47 |
| II (n = 27) | 50.4 ± 10.9 | 22 | 2.4 ± 1.6 | 18 | 12 | 10 | 3 | 232 ± 47* |
| III (n = 23) | 53.3 ± 11.0 | 15 | 2.2 ± 1.7 | 16 | 5 | 13 | 2 | 212 ± 53 |
| IV (n = 4) | 52.8 ± 10.4 | 2 | 3.3 ± 2.1 | 1 | 2 | 2 | 0 | 194 ± 9 |
| Mean ± SD | 51.4 ± 11.2 | | 2.4 ± 1.7 | | | | | 211 ± 49 |

*$p < 0.01$ (Group I versus II). Group I = aneurysm and occluded left anterior descending artery; Group II = no aneurysm and occluded left anterior descending artery; Group III = no aneurysm and patent left anterior descending artery; Group IV = aneurysm and patent left anterior descending artery; DM = diabetes mellitus; FH = family history; MI = myocardial infarction; SH = systemic hypertension; SM = smoking; TC = total serum cholesterol.

**Table 2.** Hemodynamic and Angiographic Variables

| Group | LVEDP (mm Hg) | EF (%) | Radial Shortening | | Gensini Score | | No. of Radii in IZ |
|---|---|---|---|---|---|---|---|
| | | | IZ | NIZ | Unmod | Mod | |
| I (n = 24) | 17 ± 8 | 32 ± 12* | −4.6 ± 4* | 40 ± 14 | 57 ± 14 | 94 ± 29 | 16 ± 4 |
| II (n = 27) | 16 ± 10 | 44 ± 11 | 3.1 + 4 | 38 ± 11 | 67 ± 25 | 99 ± 39 | 12 ± 4 |
| III (n = 23) | 14 ± 8 | 45 ± 15 | 3.4 + 9 | 45 ± 19 | 42 ± 33* | 59 ± 38* | 11 ± 6 |
| IV (n = 4) | 16 ± 8 | 35 ± 4* | −3.8 ± 4* | 41 ± 10 | 20 ± 9* | 56 ± 35* | 16 ± 1 |
| Mean ± SD | 16 ± 9 | 41 ± 12 | 0.5 ± 6 | 41 ± 15 | 54 ± 28 | 83 ± 36 | 13 ± 5 |

*$p < 0.001$. EF = global ejection fraction; IZ = ischemic zone; LVEDP = left ventricular end-diastolic pressure; NIZ = nonischemic zone; Unmod and Mod = unmodified and modified Gensini scores, respectively. Patient groups as in Table 1.

These differences were highly significant ($\chi^2_4 = 12.8$; $p = 0.012$) (Fig. 3). On the basis of both the unmodified and modified Gensini scores, no significant differences were found in Groups I and II. Both types of Gensini scores were significantly reduced in Groups III and IV because the left anterior descending artery was patent at the time of study in these patients (Table 2).

*The analysis of collateral blood supply* in patients with an occluded left anterior descending is shown in Figure 4. Patients without a left ventricular aneurysm (Group II) had a significantly higher score compared with patients with an aneurysm (Group I) (2.4 versus 0.9; $p < 0.001$). A direct correlation was observed between the extent of coronary artery disease (single versus multivessel disease) and collateral blood supply score (0,1 versus 2,3) in these groups ($p = 0.012$ Fisher's exact test) (Table 3). Patients with more extensive coronary disease exhibited better collateral blood supply than did those with single vessel disease.

*No group differences were observed in left ventricular end-diastolic pressure.* Both global left ventricular ejection fraction and radial shortening in the ischemic zone were significantly reduced in patients with an aneurysm (Groups I and IV) (Table 2). The number of radii in the ischemic zone and radial shortening in the nonischemic zone were similar in all four groups.

**Clinical events (Table 4, Fig. 5).** Follow-up data were available in 64 (79%) of the 79 study patients. The mean follow-up time in these patients was 48 months. No significant differences were observed in duration of follow-up and occurrence of angina pectoris, new myocardial infarction, embolic events, ventricular tachycardia or congestive heart failure. No patients were taking beta-blockers before infarction. A significantly greater percent of patients in Group II (occluded left anterior descending artery without an aneurysm) underwent coronary artery bypass surgery. The actuarial survival analysis for Groups I, II and III using a Kaplan-Meier curve was similar (Fig. 5).

**Table 3.** Relation Between Degree of Coronary Artery Disease and Degree of Collateral Blood Supply*

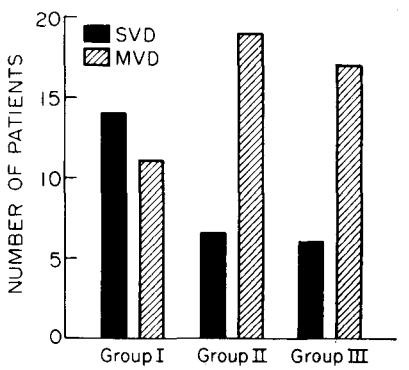
| CAD | Collateral Blood Supply | |
|---|---|---|
| | Poor (0,1) | Good (2,3) |
| Single vessel | 14 | 8 |
| Multivessel | 8 | 22 |

*Grades 0 and 1 were designated poor collateral blood supply, and Grades 2 and 3 good collateral blood supply. $p = 0.012$ (Fisher's exact test). CAD = coronary artery disease.

## Discussion

**Present study findings.** Our data demonstrate that single vessel disease is the most frequent coronary angiographic finding in patients with a left ventricular aneurysm after a first transmural anterior myocardial infarction. The extent of collateral blood supply is important in determining whether left ventricular aneurysm formation occurs in the presence of an occluded left anterior descending artery. The presence of a patent vessel, presumably due to spontaneous lysis of thrombus or resolution of spasm, was usually not associated with aneurysm formation. The extent of collateral blood supply was directly related to the extent of coronary artery disease (single versus multivessel disease). Total serum cho-

**Figure 3.** Single vessel disease (SVD) was the predominant angiographic finding in patients with an aneurysm and occluded left anterior descending artery (Group I), compared with patients without an aneurysm and occluded (Group II) and patent (Group III) left anterior descending artery. MVD = multivessel disease.



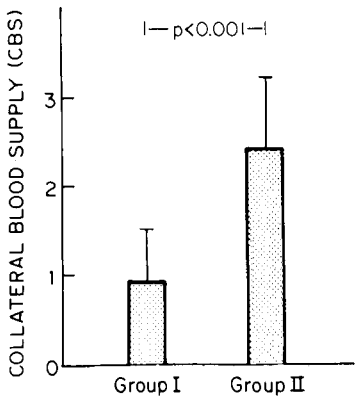



**Figure 4.** In patients with an occluded left anterior descending artery, collateral blood supply score (CBS) was significantly higher in patients without an aneurysm (Group II) compared with patients with an aneurysm (Group I) ($p < 0.001$).

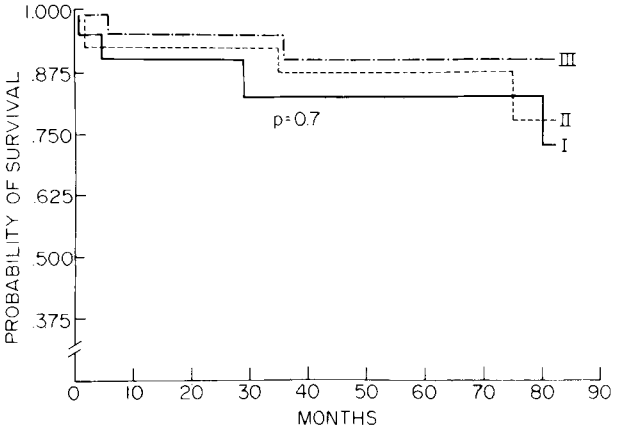



**Figure 5.** Kaplan-Meier actuarial survival curve in Groups I, II and III. No significant difference in survival is present among the three groups.

lesterol was higher in patients without an aneurysm (Groups II and III), and these patients had more extensive coronary artery disease.

*Mortality rate during the follow-up period (mean 48 months)* was low in our study, possibly because the study design had selected a group of patients evaluated a mean of 2.3 months after infarction. In addition, the fairly well preserved global left ventricular function (ejection fraction > 30%) may also have contributed to good prognosis. In accordance with previous studies (3,17), a low complication rate occurred in patients with an aneurysm. Embolic events would be expected to be low because the majority occur within 6 to 8 weeks of infarction (18) and because clinical heart failure was rare.

**Collateral blood supply.** A critical feature in determining the role of collateral vessels in the development of aneurysm is knowledge of their presence before infarction. Although our retrospective study provides no insight into this question, no studies have been reported that contain such data, and it seems highly unlikely that such data will be obtained. Current approaches to patients with a severe left anterior descending narrowing discovered before the occurrence of infarction involve interventions including intense drug therapy, percutaneous balloon angioplasty or coronary bypass surgery. Hence, it is reasonable to assume that no prospective study of the role of preexisting collaterals in the development of aneurysm will be possible.

This study, of course, cannot differentiate collateral vessels present before the infarction from those developing after infarction. However, collateral vessels may open rapidly as has been observed during balloon angioplasty and coronary spasm (19,20). In addition, this study does not provide insights into the early postinfarction aspects of aneurysm formation, because the average time from infarction to study was 2.3 months. Despite these problems related to the retrospective study design, the association between collateral circulation and aneurysm formation is clear and important. It can be hypothesized that collateral vessels protect against aneurysm formation, regardless of when or how they are formed. This is supported by the finding that angiographically visible collateral vessels in the setting of acute myocardial infarction are associated with less extensive necrosis

**Table 4.** Clinical Events During Follow-Up

| Group | Follow-Up Duration (mo) | AP | MI | EB | VT | CHF | CABG |
|---|---|---|---|---|---|---|---|
| I | 36 ± 27 | 6 | 0 | 1 | 6 | 4 | 4 |
| II | 52 ± 35 | 5 | 0 | 0 | 3 | 4 | 13* |
| III | 57 ± 25 | 7 | 0 | 0 | 1 | 3 | 9 |
| IV | 60 ± 29 | 2 | 0 | 0 | 1 | 1 | 2 |
| Mean ± SD | 48 ± 22 | | | | | | |

*$p < 0.03$. AP = angina pectoris; CABG = coronary artery bypass surgery; CHF = congestive heart failure; EB = embolic events; MI = new myocardial infarction; VT = ventricular tachycardia; patient groups as in Table 1.

and better preservation of wall motion as compared with findings in patients without collateral vessels (21–23).

**Other mechanisms involved in aneurysm formation.** Although it seems reasonable to suspect that many of the patients in our study who did not develop an aneurysm had preformed collateral vessels, such might not be so. Periinfarction phenomena not addressed in this study could play an important role in enhancing or reducing the ability to form collateral vessels. If wall tension is low, not only would myocardial oxygen consumption be reduced, with the tendency to confine the area of necrosis, but intramyocardial wall tension would be lowered and collateral vessel development potentially enhanced.

Similarly, remodeling of the left ventricle in the early postinfarction period (infarct expansion) might have a deleterious effect on collateral vessel formation. Infarct expansion has been documented in humans with both radionuclide ventriculography and echocardiography (24,25). Compensatory hyperkinesia occurring in the noninvolved segment may lead to increased wall stress, thereby promoting infarct expansion and subsequent aneurysm formation (26). Hypertension before or during infarction (James reflex) may also increase wall stress and contribute to aneurysm formation (27).

**Role of extent of coronary artery disease.** The extent of coronary artery disease associated with aneurysm formation remains controversial (1–6). Some studies (1,3,5,6) have found multivessel disease to be more common in patients who developed an aneurysm, whereas others (2,4) have found a preponderence of single vessel disease. Less than one-third of patients in the CASS study had single vessel disease compared with nearly half of the patients studied by Faxon et al. (1) and Rogers et al. (2). The presence of prior infarction and the variable time to catheterization may have influenced these results. Our data demonstrate that single vessel disease is the most prevalent angiographic finding in patients developing an aneurysm after a *first* transmural anterior infarction studied early (mean of 2.3 months) after infarction.

**Conclusions.** Total occlusion of the left anterior descending artery in association with inherent poor collateral blood supply predisposes to left ventricular aneurysm formation after anterior myocardial infarction. Multivessel disease with good collateral blood supply or a patent left anterior descending artery is uncommonly associated with the development of an aneurysm. The degree of collateral blood supply appears to be directly related to the overall severity of coronary disease. This study substantiates the importance of residual blood flow to the infarct zone whether present by anterograde flow in the infarct vessel (with or without spontaneous reperfusion) or by way of collateral blood supply. Patients with single vessel disease and poor collateral circulation may benefit from interventions to reestablish flow in the occluded bed even at a time when the amount of salvagable myocardium is small, with the reasonable expectation that aneurysm formation would be prevented.

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